AD-A128 154 HYPOHYDRATION AND ACCLIMATION: PLASMA RENIN ACTIVITY AND ALDOSTERONE DURI..(U) ARMY RESEARCH INST OF ENVIRONMENTAL MEDICINE NATICK MA 1/1

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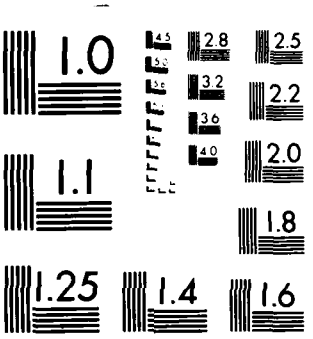

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This study was designed to assess the effects of hydration, acclimation, and environment on the response of fluid regulatory hormones to exercise. Sixteen subjects exercised (1.34 $m.sec^{-1}$), both pre-and post-acclimation, when euhydrated or hypohydrated (-5% of body weight) in a comfortable (20°C, rh - 40%), hot-wet (35°C, rh = 79%), or hot-dry (49°C, rh = 20%) environment. While light exercise in a thermoneutral environment had no effects on plasma levels of renin activity (PRA) or aldosterone (ALD), exercise in both hot environments resulted in significantly increased levels of these hormones. Increments in both

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PRA and ALD were greater when hypohydrated, and PRA effects were significantly moderated by heat acclimation in both the euhydration and hypohydration experiments. While PRA and ALD responses were generally correlated, acclimation did not consistently attenuate ALD increments. We concluded that hydration state, acclimation level, and environmental conditions all affected the responses of these hormones to light exercise.

Hypohydration and acclimation: plasma renin activity and aldosterone during heat/exercise stress

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A

Abstract

This study was designed to assess the effects of hydration, acclimation, and environment on the response of fluid regulatory hormones to exercise. Sixteen subjects exercised (1.34 m/sec^{-1}), both pre- and post-acclimation, when euhydrated or hypohydrated (-5% of body weight) in a comfortable (20°C, rh = 40%), hot-wet (35°C, rh = 79%), or hot-dry (49°C, rh = 20%) environment. While light exercise in a thermoneutral environment had no effects on plasma levels of renin activity (PRA) or aldosterone (ALD), exercise in both hot environments resulted in significantly increased levels of these hormones. Increments in both PRA and ALD were greater when hypohydrated, and PRA effects were significantly moderated by heat acclimation in both the euhydration and hypohydration experiments. While PRA and ALD responses were generally correlated, acclimation did not consistently attenuate ALD increments. We concluded that hydration state, acclimation level, and environmental conditions all affected the responses of these hormones to light exercise.

Key words: Exercise; hot-dry; hot-wet; temperate; acclimation; dehydration; plasma hormones.

Introduction

The role of fluid regulatory hormones in the acquisition of heat acclimation has been investigated for a number of years. As early as 1967 Braun et al. (1) attempted to accelerate the acclimation process in men by the administration of d-aldosterone. Although their subjects demonstrated several beneficial effects of hormone treatment (1), no reduction in total acclimation time was achieved. Finberg et al. (6) reported that increments in plasma renin activity induced by exercise/heat stress were attenuated when the subjects were heat acclimatized and euhydration was maintained. In a subsequent experiment these investigators (5) demonstrated that a 7-day heat acclimation program significantly reduced the increments in plasma renin activity during exercise in the heat, but plasma aldosterone was unaffected. However, Davies et al. (3) reported that increases in plasma renin activity and aldosterone during exercise in the heat were unaffected by heat acclimation; saline consumption reduced, but did not prevent, these increments. Convertino et al. (2) found that both sedentary heat exposure and cycle ergometry at a moderate temperature induced significant increases in plasma renin activity; consecutive exposures to either condition did not modify the increments in plasma renin activity.

Recently, Gaebelein and Senay (9) have suggested that exercise-induced changes in serum osmolality may be dependent not only upon the mode of exercise, but also the initial hydration level of the test subjects. Additionally, Senay (17) had earlier reviewed data indicating that the training level of test subjects might also be expected to affect body fluid responses to exercise in a hot environment. Thus, it is evident that state of acclimation, hydration level, exercise mode, and physical condition may independently or collectively affect the direction and intensity of body fluid shifts or hormonal responses during exercise in a hot environment. In addition, we cannot rule out the possibility

that other factors might inf[illegible]uch responses. In the current experiment we have elected to investigate [illegible]veral interactive variables reported or hypothesized to alter hormonal r[illegible]nses to exercise in the heat.

Since plasma renin activity (PRA) has been correlated with plasma aldosterone (ALD) levels (10, 13) under a variety of conditions including exercise (8), heat acclimation (3), and heat stress (11), we have examined the responses of both hormones to exercise in the heat. Because hypohydration might be expected to affect these alterations, we have also opted to examine the impact of reduced fluid reserves on these responses. Further, the effects of heat acclimation and environmental conditions were simultaneously investigated to obtain a comprehensive profile on the control of these adaptive endocrine responses.

Methods

Eight male and 8 female test volunteers (Ss) participated in this study; male test subjects had a mean age (±SD) of 23.6 ± 2.8 years, height of 170.8 ± 7.2 cm, and weight of 75.4 ± 7.4 kg. Respective data for the women were 25 ± 4.2 years, 163.0 ± 6.6 cm, and 62.2 ± 11.3 kg. Before initiation of the study each subject was fully apprised of the rationale, methods, procedures, and potential risks of the study. Each volunteer reserved the right to withdraw at any time without retribution.

Each subject participated in a total of 12 tests, 6 before and 6 after completion of a heat acclimation program. Prior and subsequent to acclimation Ss completed two experimental tests in each of three environments: thermoneutral (Ta = 20°C, rh = 40%), hot-wet (Ta = 35°C, rh = 79%), and hot-dry (Ta = 49 C, rh = 20%). The tests were repeated twice in each environment - once when each volunteer was euhydrated and once when hypohydrated. Each

experimental test comprised a total of 140 min (4 repeated intervals of 10 min rest and 25 min exercise). Exercise was performed on a level treadmill at 1.34 $m \cdot s^{-1}$; during each rest period Ss were weighed and rehydrated with cool tap water to maintain either baseline weight or -5% from baseline depending upon the euhydrated or hypohydrated condition.

Hypohydration was accomplished by voluntary restraint from fluid consumption for 24 h before a test and also by performing mild exercise in a hot (Ta = 38°C, rh = 20%) environment until 5% of initial body weight was lost. Having achieved the appropriate weight loss, Ss were removed to a comfortable environment and spent the night under supervision. Ss were awakened at 0600h, weighed, provided a light breakfast if their weight was sufficiently low, and tested at approximately 0800h.

The acclimation program consisted of 10 consecutive days of walking on a level treadmill at 1.34 $m \cdot s^{-1}$ for two 50 min exercise bouts interrupted by a 10 min rest period. Environmental conditions were alternated (i.e. 1st day, hot-dry as above; 2nd day hot-wet, etc.). During the acclimation and the exercise test intervals, Ss wore shorts, t-shirts, and tennis shoes; ad lib water was encouraged during the acclimation period.

On each of the test days venous blood (5 ml) was obtained by a catheter placed in a superficial arm vein. The first blood sample (time 0) was taken after the Ss stood for 20 min in a moderate environment (Ta = 20°C, rh = 30%); the second (time 1) and third (time 2) samples were removed at approximately 15-20 min of the the first and second exercise bout in the appropriate environment. The final (time 4) blood sample was obtained at the completion of the fourth exercise bout or at the completion of exercise if the S was unable to complete the entire protocol. Blood was collected without stasis, transferred to iced, heparinized tubes, centrifuged (10000 g, 4°C), and the plasma removed and frozen (-20°C) for subsequent assay.

Plasma samples were thawed and PRA and ALD levels were quantitated by radioimmunoassay using commercially available test kits manufactured by International CIS (Saluggia, Italy) and distributed by Damon Diagnostics (Needham, MA). Angiotensin I in the PRA assay procedure was generated at pH 6.0 (37°C) for 1 h. Both assays were performed according to standard techniques described in the respective technical bulletins.

Student's t test for paired data were performed on appropriate paired samples as noted in the Results section. When values at each time interval were consecutively compared with a single control, Dunnett's t test was employed (12). The null hypothesis was rejected at $p < .05$.

Results

Results indicated that there were no effects of gender on hormonal responses; thus, data were combined under all conditions. Fig. 1 demonstrates the effects of hypohydration and acclimation on PRA during exercise in the thermoneutral environment. In the pre-acclimation samples, hypohydration resulted in a significant ($p < .05$) increment at each observation; however, in the same samples no effects of exercise were noted in this comfortable environment under either hydration condition. Following acclimation, PRA was significantly increased at time 0 ($p < .01$) and time 4 ($p < .001$) in the hypohydrated condition (vs. euhydrated). It is also interesting to note that, following acclimation, exercise in this thermoneutral environment elicited a significant decrement ($p < .05$) in PRA in the time 4 (vs time 0) sample during euhydration and significant ($p < .05$) decrements at times 1,2, and 4 (vs time 0) during hypohydration. Heat acclimation resulted in reduced PRA in both the euhydrated (time 4) and hypohydrated (times 1,2,4) states.

Fig. 2 depicts the effects of hypohydration and acclimation on PRA during exercise in a hot-wet environment. Once again, in the euhydrated state and preacclimation, PRA was significantly ($p < .05$) less than the corresponding value in the hypohydrated condition. However, in this hot-wet environment it is apparent that exercise resulted in significant ($p < .05$) increments at times 1,2, and 4 (vs time 0) in the euhydrated state. In the hypohydrated condition, a significant ($p < .05$) increment is observed at the time 4 (vs time 0) interval only. Heat acclimation greatly modified the response of PRA to exercise in the hot-wet environment. For example, following acclimation in the euhydration trial exercise elicited no significant differences (time 0 vs time 1,2, or 4) while during hypohydration only at time 4 (vs time 0) was the increment significant ($p < .05$). During the hypohydration test acclimation caused a significant decrement at each sampling interval (pre- vs post-acclimation).

Fig. 3 illustrates the effects of hypohydration and acclimation on PRA during exercise in a hot-dry environment. Once again, pre-acclimation, hypohydration elicited significantly ($p < .05$) increased levels of PRA at each sampling interval. Pre-acclimation, exercise elicited significant ($p < .05$) increments at times 2 and 4 (vs time 0) in both the euhydrated and hypohydrated states. Following acclimation exercise effected significant ($p < .05$) increments (time 0 vs time 1,2, or 4) in both the eu- and hypohydrated state. The effects of acclimation were most apparent in the hypohydrated condition with significant ($p < .01$) reductions noted in exercise responses at each sampling time.

Figs. 4-6 summarize the effects of hypohydration, acclimation and heat stress on plasma aldosterone (ALD) during exercise. In the thermoneutral environment, pre-acclimation, (Fig. 4) hypohydration effected a significant ($p < .05$) increment in plasma ALD levels. However, mild exercise in this environment had no effects on ALD levels, whether Ss were eu- or hypohydrated.

Following acclimation, hypohydration elicited significant (e.g. time 4, post-acclimation, hypohydrated vs euhydrated, $p < .005$) increments in plasma ALD levels. Of considerable interest is the observation that following acclimation when euhydrated, exercise in the thermoneutral environment elicited a pattern of decreasing levels of ALD culminating in a significant ($p < .05$) decrement in the final (time 4 sample) when compared to the pre-exercise value (time 0). This did not occur in the hypohydration test nor were any further significant effects of acclimation noted.

Fig. 5 depicts the effects of hypohydration and acclimation on plasma ALD responses during exercise in a hot-wet environment. Once again, in the pre-acclimation samples at each time interval, hypohydration effected significant ($p < .005$) increments in levels of ALD. Exercise in the hot-wet environment elicited significant increments ($p < .05$) in ALD levels in both the euhydrated (time 0 vs times 2, 4) and the hypohydrated (time 0 vs times 1,2,4) conditions. Following acclimation the effects of hypohydration on plasma ALD were negated while exercise again elicited significant increments in the euhydrated (time 0 vs times 2,4) and hypohydrated (time 0 vs times 1,2,4) states. In the euhydrated condition following acclimation there occurred significant ($p < .001$) increments in plasma ALD (times 0,1,2-pre-acclimation vs post-acclimation) levels.

Finally, Fig. 6 demonstrates the effects of hypohydration and acclimation on plasma ALD alterations during exercise in a hot-dry environment. Pre-acclimation, hypohydration elicited significant ($p < .005$) increments at each sampling time. Further, exercise in the hot, dry environment, pre-acclimation, evoked significant ($p < .05$) increments at all times (time 0 vs times 1,2,4) in the euhydrated state and at times 2,4 (vs time 0) in the hypohydrated. Following acclimation, hypohydration again effected significant increments in plasma ALD levels ($p < .05$, times 1,2,4) while exercise again produced significant effects in

both the eu- (time 0 vs times 2,4) and hypohydrated (time 0 vs times 1,2,4) conditions. In this hot-dry environment acclimation had no effects on plasma ALD levels in either eu- or hypohydration.

Discussion

The results of this investigation demonstrated that: 1. hypohydration elicited increased levels of PRA and ALD in both acclimated and non-acclimated individuals; 2. the light exercise intensity employed in this study e :cted increments in PRA and ALD in conjunction with both hot environm ., 3. acclimation to heat reduced levels of PRA in both euhydrated and hypoh . d Ss; 4. acclimation to heat did not consistently affect responses of plasma ALD particularly in euhydrated volunteers; 5. mean PRA and ALD levels were highly correlated ($r = .80$, $p < .005$); and 6. gender did not affect PRA and ALD responses to heat/exercise stress.

To our knowledge this is the first study which has evaluated the effects of hypohydration, as well as acclimation, on the response of fluid regulatory hormones of female and male test volunteers to exercise in the heat. The large number of samples processed enabled us to evaluate the effects of these parameters and draw conclusions as to the role of the fluid regulatory hormones in these processes and under these conditions. It is interesting to note that prior to acclimation, and before exercise (i.e. time 0 samples), hypohydration had marked incremental effects on circulating levels of PRA (112%, 135%, 214%) and ALD (61%, 72%, and 101%) respectively, in the temperate, hot-wet, and hot-dry experiments. Following acclimation these elevations were reduced to 87.5%, 8.6% and 51% for PRA and 35%, 0%, and 15% for ALD. These results are consonant with the hypothesis that hypohydration following acclimation impacts less upon fluid regulatory hormones due to the increased fluid volume associated

with heat acclimation. Also, prior to heat acclimation, exercise in either the hot-wet or hot-dry environment elicited significant increments in levels of both PRA and ALD when hypo- or euhydrated. Again, in both conditions (eu- or hypohydration) acclimation moderated these responses. Although fluid was replaced during the time of the actual exercise protocol in the current experiments, Myhre and Robinson (14) had demonstrated (during sedentary heat exposure) that plasma volume was decreased by 2.9% in 6 Ss at 50°C even when euhydration was maintained. During mild dehydration (2.6%) the same workers (14) demonstrated a 7.8% reduction in plasma volume. In our experiments the degree of hypohydration was even greater, and this was clearly manifested in the hormonal responses attained preacclimation during hypohydration.

Generally, some of our findings are similar to those reported by Finberg and Berlyne (5). Although conditions between the two studies differed considerably, we have confirmed that heat acclimation attenuated the response of PRA to exercise in the heat while plasma ALD responses were less affected by acclimation. Despite several specific differences in PRA and ALD responses to exercise in the heat, PRA and ALD levels were generally correlated. The reduced response of PRA following heat acclimation may be partially attributed to the increased plasma volume accompanying acclimation (10,16), attenuated renal vasoconstriction subsequent to acclimation (15), or an overall decrease in sympathetic response to exercise in the heat following acclimation (4).

While there were no notable differences between the effects of the hot-wet and hot-dry environments, the results clearly indicated that light exercise in the thermoneutral environment did not evoke an endocrine response. Usually, the intensity of the hormonal response appears to be related to the severity of the heat stress. For example, Follenius et al. (7) demonstrated minor increments in levels of PRA and ALD when salt-repleted Ss were exposed for 90 min to a

heat stress of 46°C. Kosunen et al. (11) reported 2- and 5-fold elevations in PRA and ALD, respectively, upon sedentary exposure for 20 min to 85-90°C. From the current experiments we concluded that light exercise in the thermoneutral environment created neither an exercise nor an environmental stress of sufficient magnitude to elicit an endocrinological response. Further, since Ss were acclimated to both the hot-wet and hot-dry environments, it is not surprising that PRA and ALD responses were similar under both conditions during exercise in the heat. Likewise, the effects of heat acclimation in modulating the heat/exercise response of PRA were comparable under both heat conditions.

We have concluded from these experiments that while hypohydration is extremely effective in elevating plasma PRA and ALD levels, heat acclimation greatly moderates PRA responses, but has a smaller effect on ALD alterations. Further, mild exercise in a thermoneutral environment had no effects on PRA or ALD while imposition of a hot-wet or hot-dry environment was equally effective in inducing elevated levels. Finally, while PRA and ALD alterations were generally correlated, analogous responses were not consistently observed. All three variables - hypohydration, acclimation, and environment - affected hormonal responses. It would be useful to extend these studies to include other heat intensities and levels of hypohydration to determine whether hormonal responses are modulated by varying conditions. Interindividual differences in hormonal responses might ultimately be related to the level of acclimation achieved or the ability to withstand heat/exercise/hypohydration stress.

Acknowledgements and Disclaimers

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The views of the authors do not purport to reflect the positions of the Department of the Army or the Department of Defense.

Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

Figure Legend

Fig. 1 Effects of hypohydration and acclimation on plasma renin activity during exercise in a thermoneutral environment. Mean values $\pm$ SEM are depicted for n = 16 in all cases except n = 15 pre-acclimation, hypohydration, times 2 and 4. Blood samples were removed after standing for 20 min (time 0), 15-20 min of the first and second exercise bouts (times 1 and 2) and at the end of exercise (time 4). Exercise was performed at 1.34 $m \cdot sec^{-1}$, level treadmill at an environmental temperature of 20°C and humidity of 40%.

Fig. 2. Effects of hypohydration and acclimation on plasma renin activity during exercise in a hot-wet environment. All conditions are as noted under Fig.1 except Ta = 35°C, rh = 79% and n = 15, pre-acclimation, euhydrated time 4 and n = 14, post-acclimation, hypohydrated, time 4.

Fig.3. Effects of hypohydration and acclimation on plasma renin activity during exercise in a hot-dry environment. All conditions are as noted under Fig. 1 except Ta = 49°C, rh = 20% and n = 14, pre-acclimation, hypohydrated, time 4.

Fig. 4. Effects of hypohydration and acclimation on plasma aldosterone levels during exercise in a thermoneutral environment. All conditions are as noted under Fig. 1.

Fig. 5. Effects of hypohydration and acclimation on plasma aldosterone levels during exercise in a hot-wet environment. All conditions are as noted under Figs. 1 and 2.

Fig. 6. Effects of hypohydration and acclimation on plasma aldosterone levels during exercise in a hot-dry environment. All conditions are as noted under Figs. 1 and 3.

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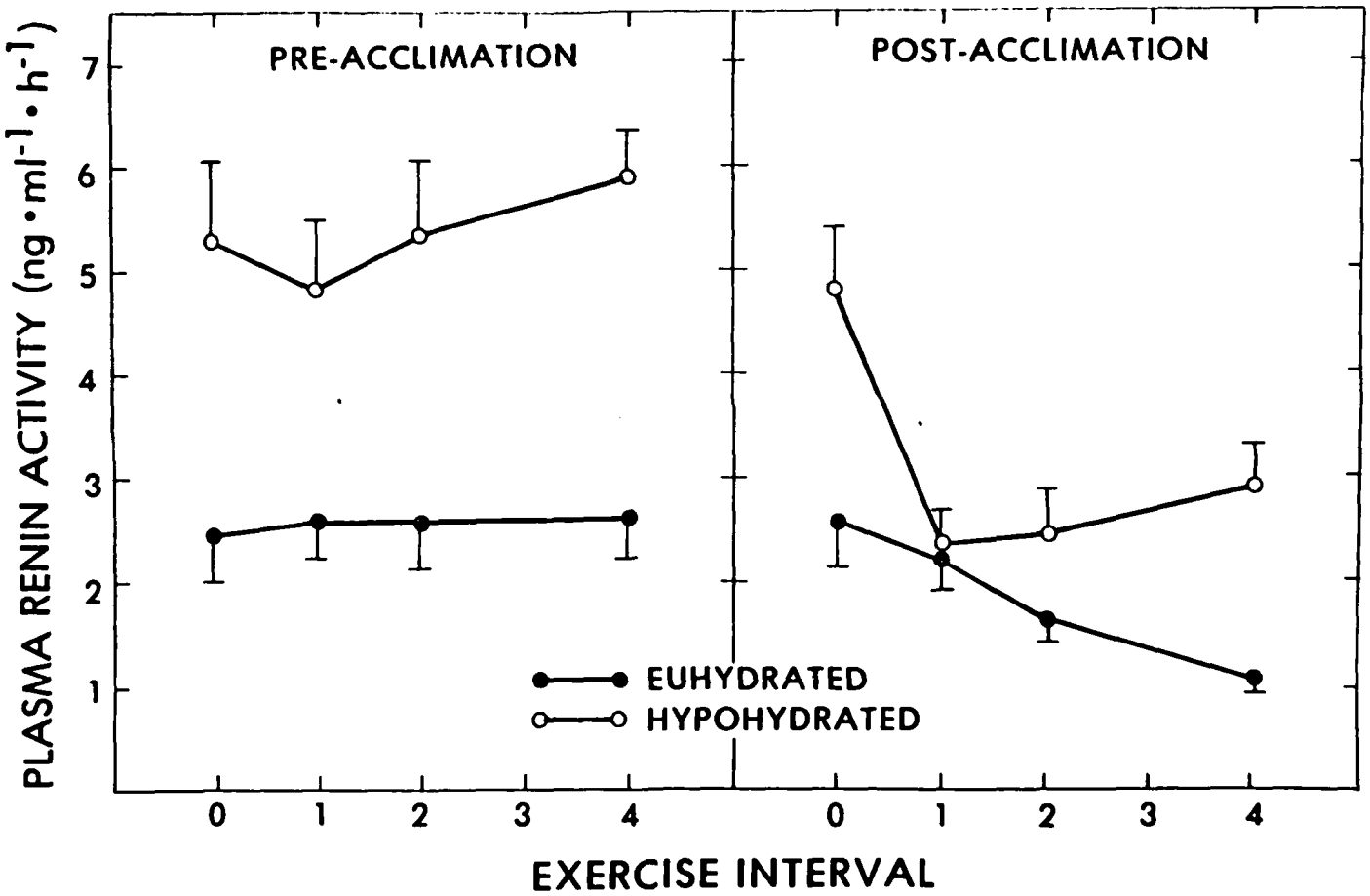
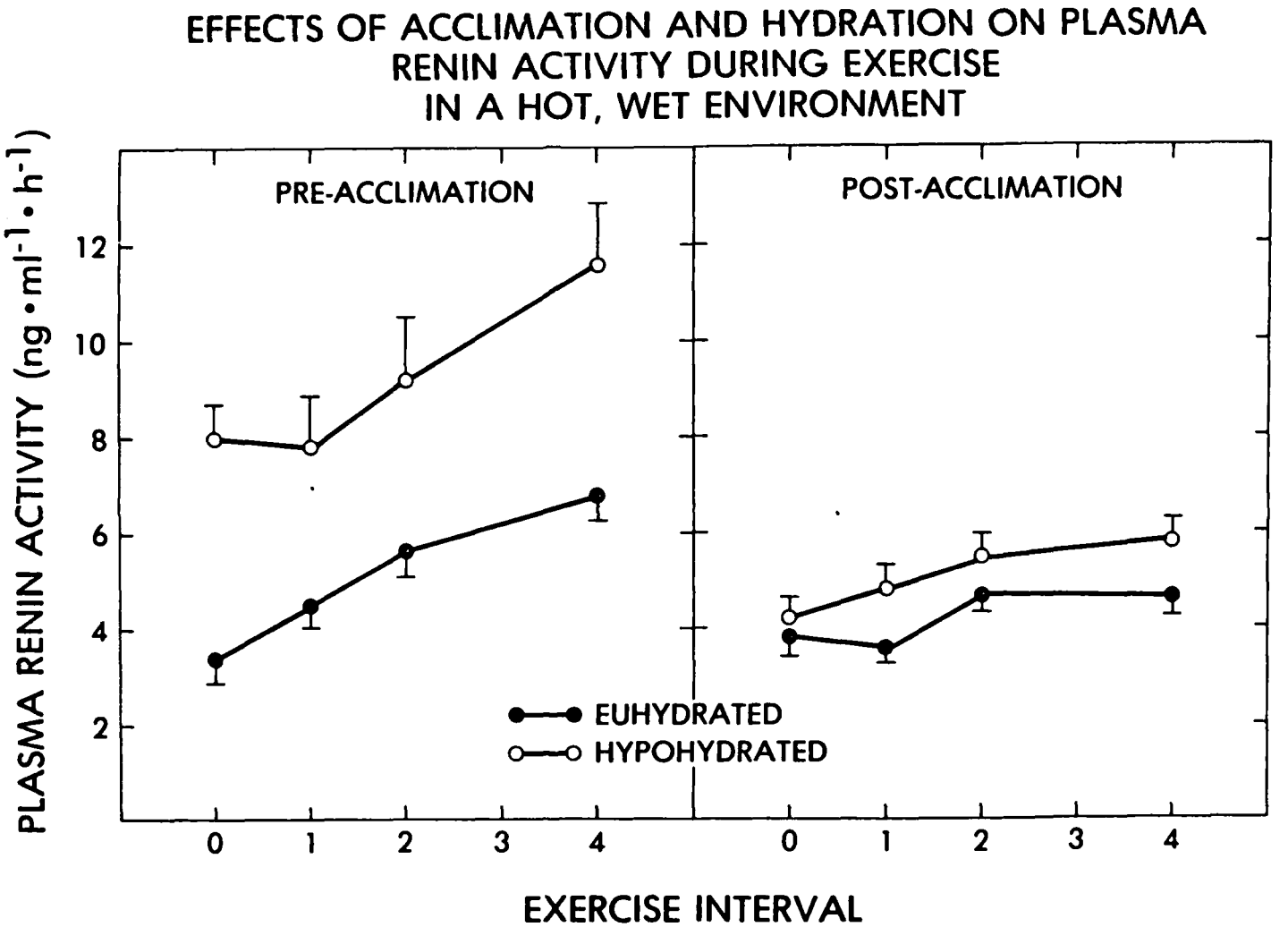
EFFECTS OF ACCLIMATION AND HYDRATION ON PLASMA RENIN ACTIVITY DURING EXERCISE IN A THERMONEUTRAL ENVIRONMENT

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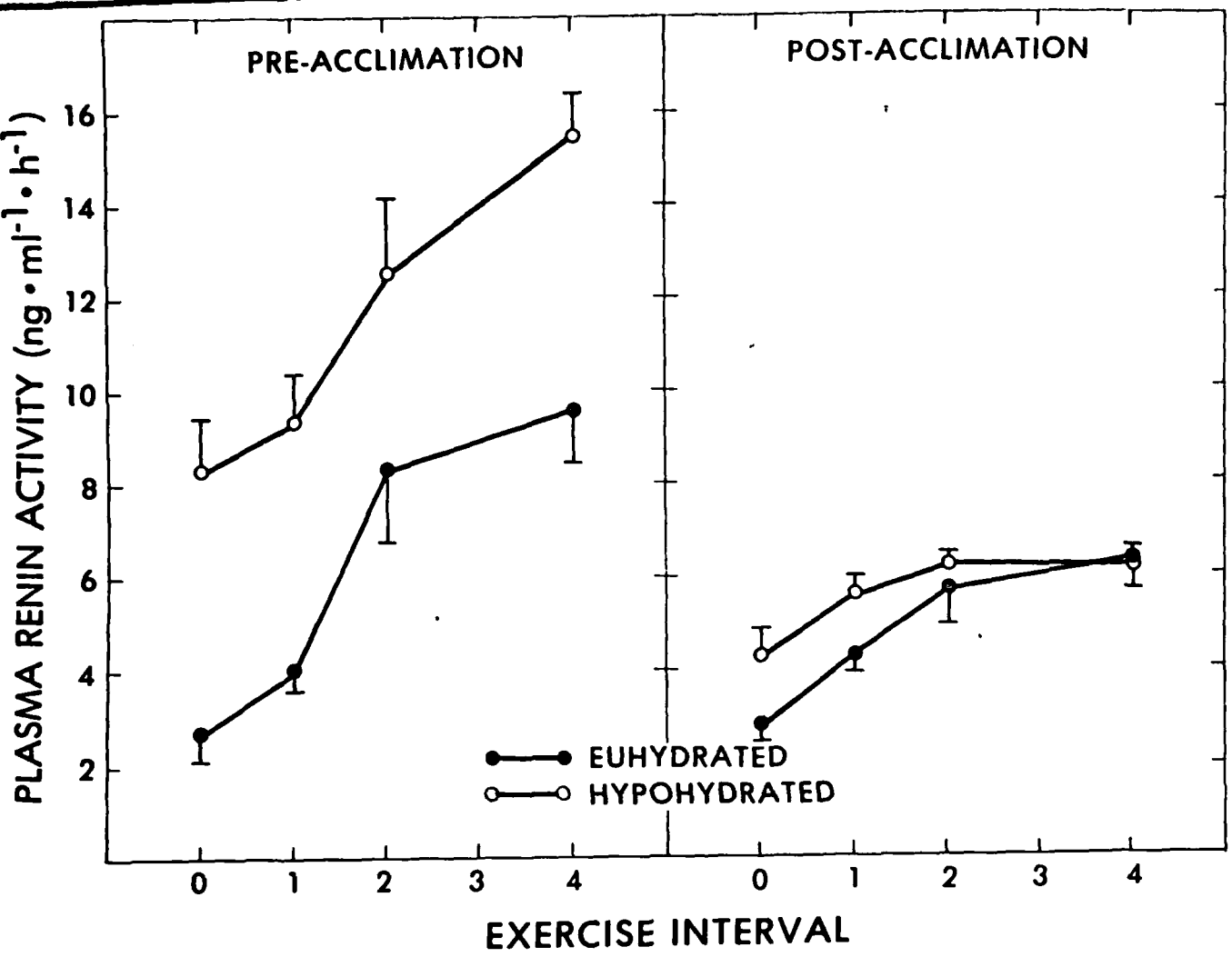
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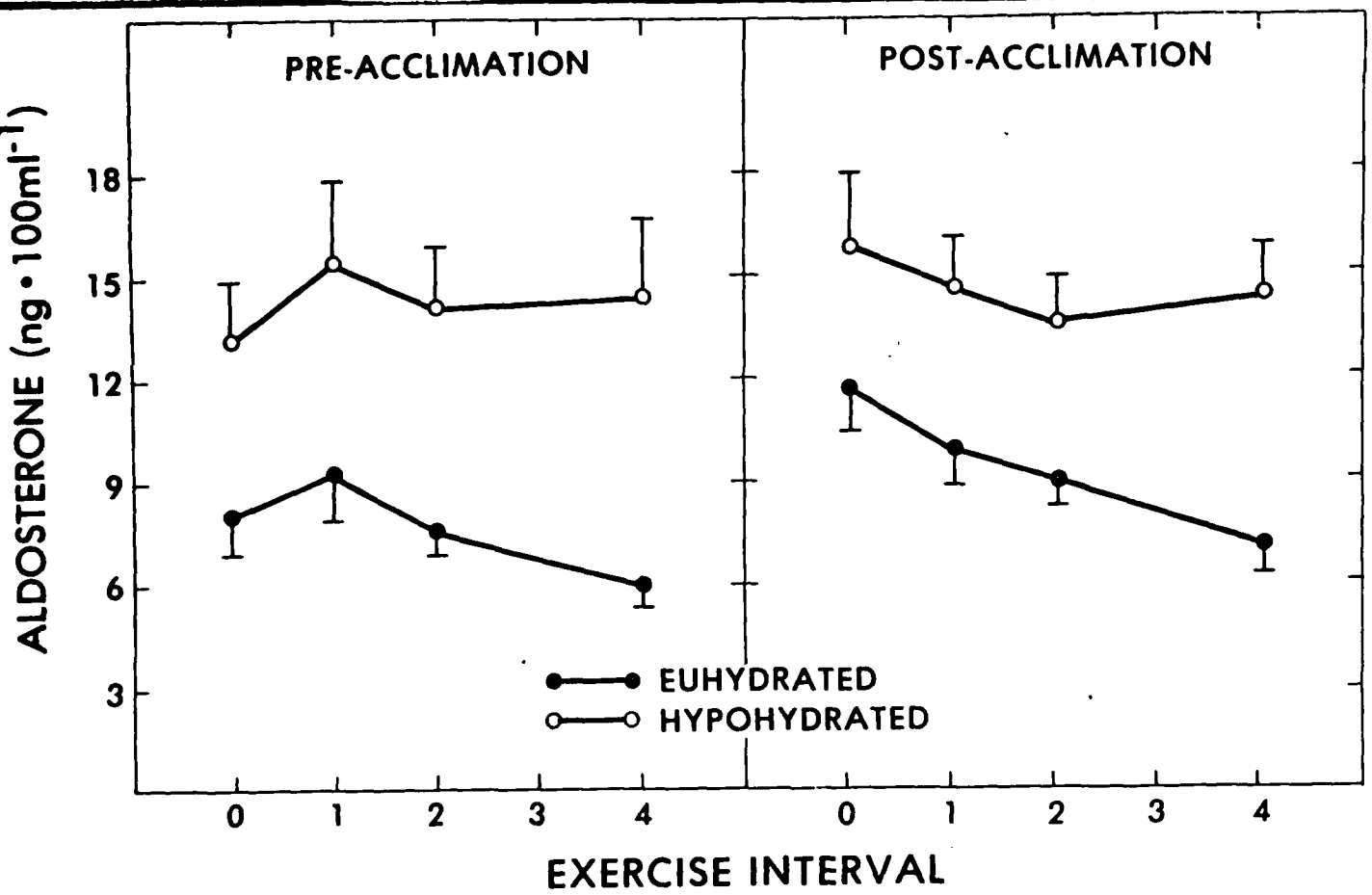
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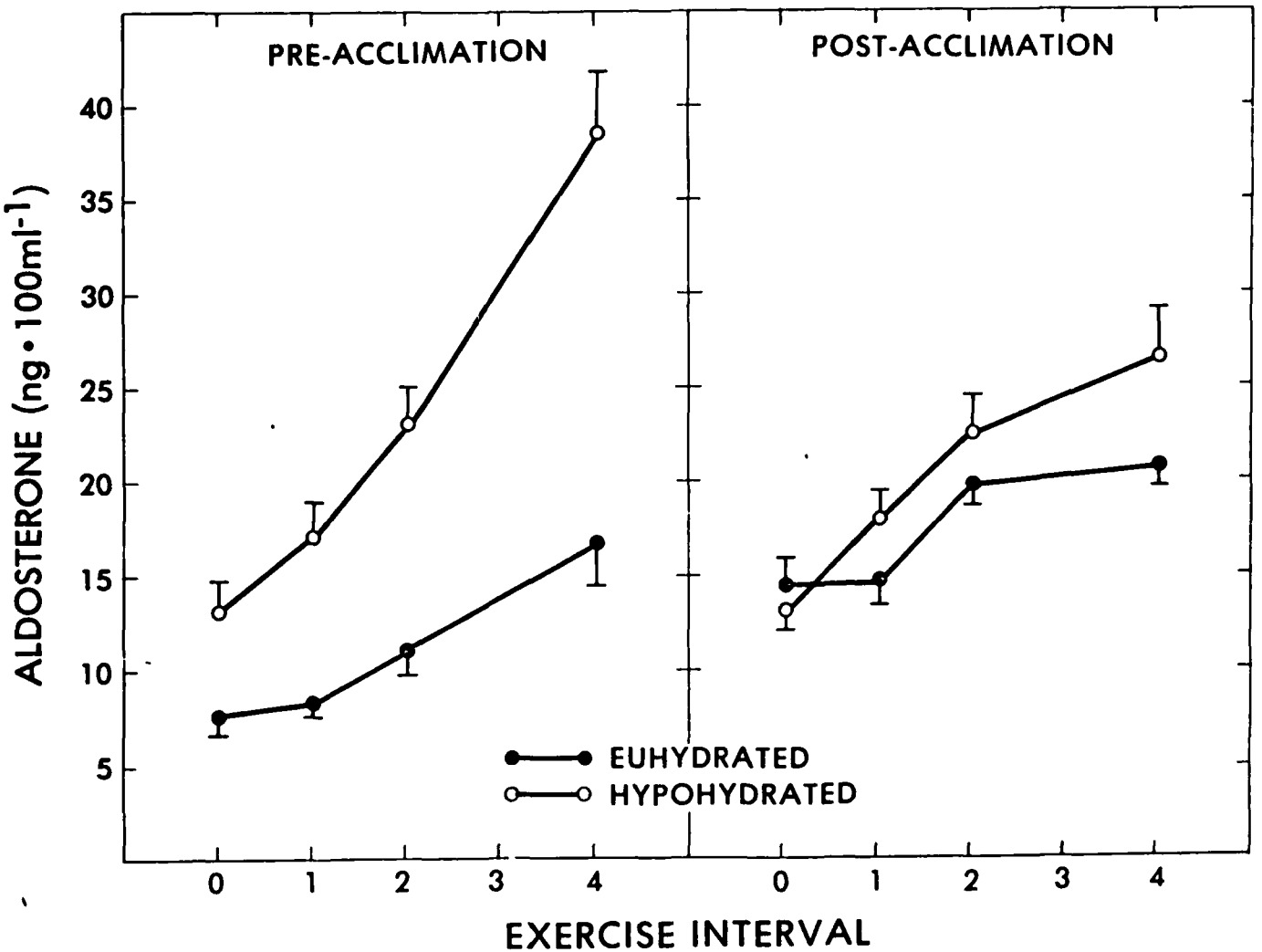
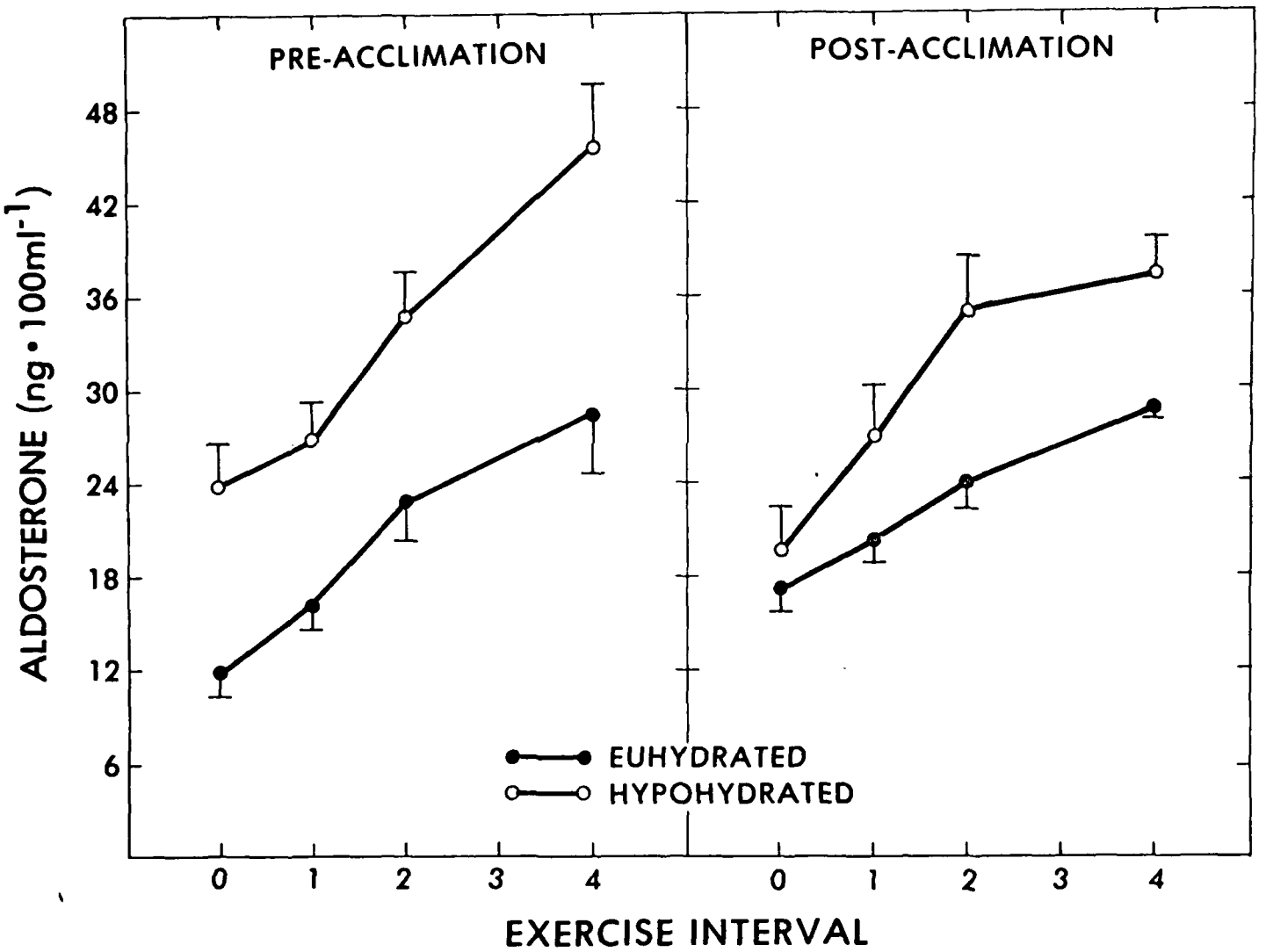
EFFECTS OF ACCLIMATION AND HYDRATION ON PLASMA ALDOSTERONE LEVELS DURING EXERCISE IN A HOT, WET ENVIRONMENT



EFFECTS OF ACCLIMATION AND HYDRATION ON PLASMA ALDOSTERONE LEVELS DURING EXERCISE IN A HOT, DRY ENVIRONMENT




MEL
8